CLINICAL STUDY OF VARICOSE VEINS OF LOWER LIMB

DISSERTATION SUBMITTED FOR

BRANCH – I

M.S. (GENERAL SURGERY)

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CERTIFICATE

This is certify that this dissertation entitled **"CLINICAL STUDY OF VARICOSE VEINS OF LOWER LIMB"** submitted by **Dr.N.DEEPA** to the TamilNadu Dr.M.G.R Medical University, Chennai, is in partial fulfillment of the requirement for the award of M.S Degree Branch – I (General Surgery) and is a bonafide research work carried out by her under direct supervision and guidance.

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DECLARATION

This is a consolidated report on "**CLINICAL STUDY OF VARICOSE VEINS OF LOWER LIMB"** based on 75 cases treated at Govt. Rajaji Hospital, Madurai, during the period of July 2004 to September 2006.

This is submitted to the **Tamilnadu Dr.M.G.R. Medical University, Chennai** in partial fulfillment of the rules and regulations for the **M.S.** Degree Examination in General Surgery.

It was not submitted to the award of any degree/diploma to any university either part or in full form previously.

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Madurai Medical College, **DR. N.DEEPA**

Madurai.

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At the outset, I wish to thank our Dean **Dr.S.M. Sivakumar MS,** for permitting me to use the facilities of Madurai Medical College and Govt. Rajaji Hospital to conduct this clinical study on varicose veins of lower limbs.

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Last but not the least; I thank all the patients for their kind cooperation in carrying out the study successfully.

CONTENTS

INTRODUCTION

Varicose veins refer to any dilated, tortous, elongated vein of any caliber. The term varicose veins is, in the common parlance, a term that encompasses a spectrum of venous dilation that ranges from minor telangiectasia to severe dilated veins. Telangiectasias are intra dermal varicosities that are small and tend to be cosmetically unappealing but not symptomatic. Reticular veins are subcutaneous dilated veins that enter the tributaries of the main axial or trunk veins.

Varicose veins of lower limbs are the penalty, man has to pay for his erect posture. They are associated with high morbidity even though mortality may not be significant. Twenty percent of the population suffer from varicose veins. High rate of recurrence after surgery, difficulty in surgery warranty thorough clinical examination, complete investigation and optimal treatment.

Hippocrates recognized an association between varicose veins and leg ulcers. He was the first person to advocate the use of compression bandages to treat leg ulcers.

Homans attributed leg ulceration to hypoxia. The presence of a fibrin cuff and was thought to impede oxygen transfer.

Professor Michel was the first to recognize the trapping of white cells in dependent limb.

Coleridge smith, Thomas Dormandy and Scurr proposed the Hypothesis that white cell trapping was important in venous ulceration.

AIM OF THE STUDY

1. To study the incidence of varicose veins in relation to
    a. Venous Ulceration
    b. Age
    c. Sex
    d. Occupation

    Of the patients admitted in Government Rajaji Hospital, Madurai.

2. To study the various etiological factors for varicose veins.
3. To study the familial incidence of varicose veins.
4. To study the clinical patterns of varicose veins and comparing their occurrence with etiological factors.
5. To study the incidence of various venotensive changes and complications.
6. To study the relevance of various investigative modalities for varicose veins (particularly those available at G.R.H. Madurai).

MATERIALS AND METHODS

Admitted patients in Government Rajaji Hospital, Madurai were taken up for study with

a) Through history taking
b) Clinical examination for pattern of varicose veins, etiological factors, source and pathway of varicose veins, venotensive changes and complications.
c) Results of investigative modalities like Duplex Doppler Study, phlebography and ultrasonography that are available in G.R.H., Madurai.

ANATOMY OF LOWER LIMB VEINS

Deep veins

Deep veins of lower limb accompany the arteries and their branches. Main veins are posterior tibial vein and its tributaries, peroneal vein, anterior tibial vein, popliteal vein and femoral vein.

The characteristic features of deep veins are;

1. There are numerous valves in these veins. These valves direct the flow of the blood upwards and prevent regurgitation of blood.
2. All the muscles of lower limbs ranging from diminutive muscles like plantaris to massive muscles of calf have numerous venous sinuses. These sinuses communicate to deep veins and through perforator veins to superficial veins. Perforators have valves and these direct blood from superficial veins to sinuses and then to deep veins.

Superficial veins

They are long and short saphenous veins and their tributaries and they lie in subcutaneous fat.

Long saphenous vein start from dorsal venous arch of foot at medial end, course in front of medial malleolus, in medial aspect of leg, medial to knee, in medical aspect of thigh and passes thro’ fossa ovalis to end in femoral

vein. It's main tributaries are posterior arch vein (arcuate vein) and anterolateral vein in leg, posteromedial, anterolateral, superficial epigastric, external pudendal and circumflex iliac veins in thigh. Below knee it is closely related to saphenous nerve.

Short saphenous vein starts in lateral end of dorsal venous arch, runs in lateral aspect of leg, enters deep fascia at a variable level between knee and midcalf and joins popliteal vein at variable level but usually opposite femoral condyles. It's upward extension known as Giacomini vein may run deeply in continuity with profunda femoris vein or superficially curving round to join posteromedial vein of long saphenous group.

Superficial veins also have multiple valves which allow only upward blood flow and their number is more in lower part of leg.

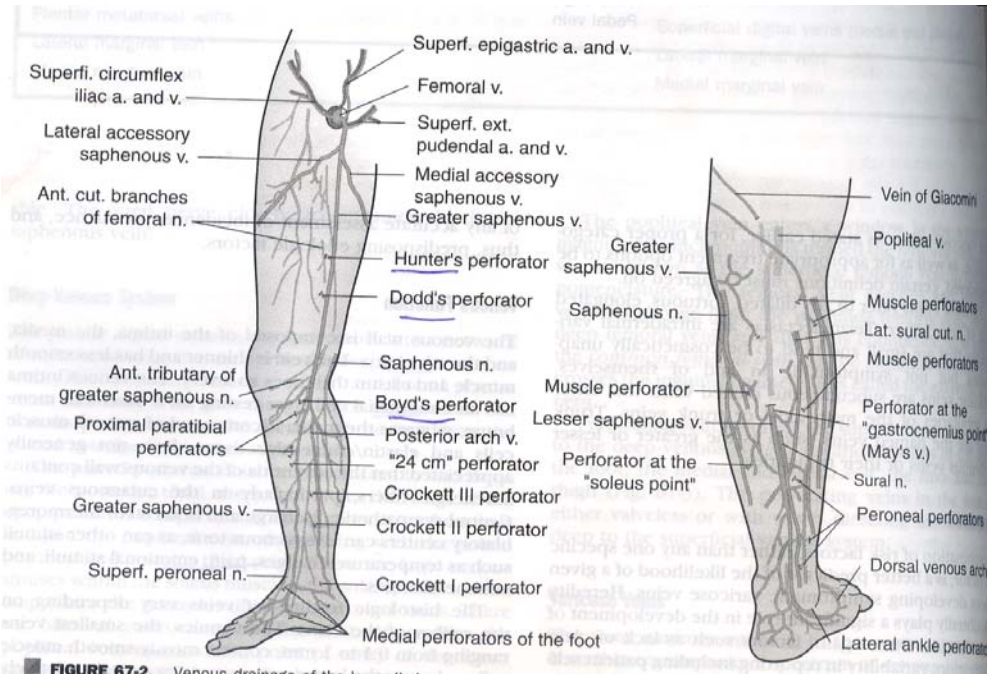
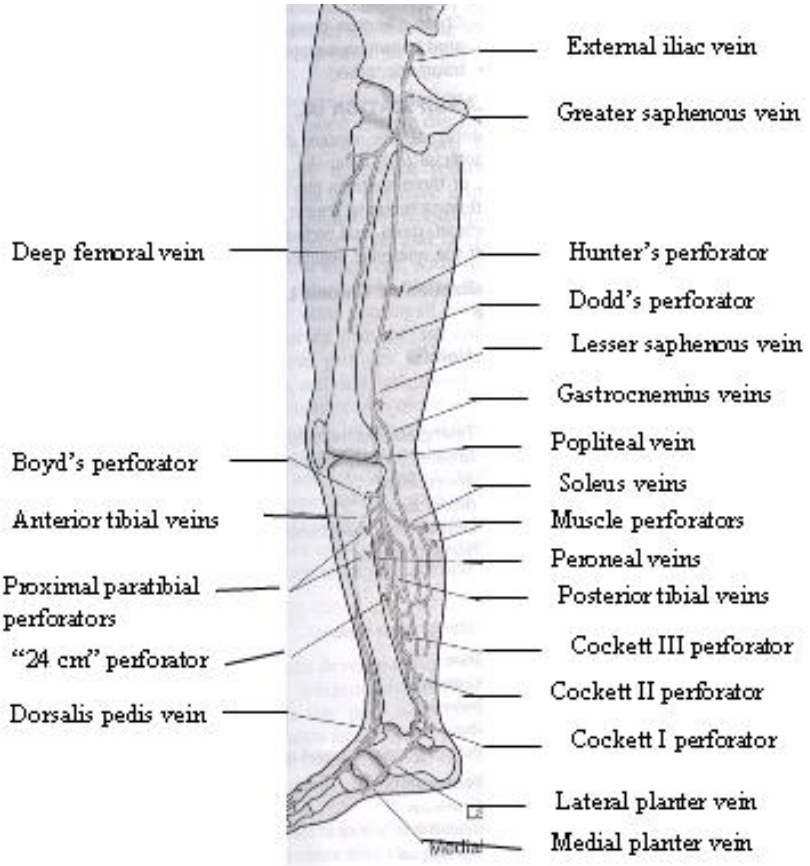


FIGURE 67-2

VEINS OF THE LOWER LIMB

Perforating veins

These veins connect superficial veins to either deep veins or venous sinuses of leg muscles. They are valved allowing only inward blood flow and their number is more than 60. The arrangement of fibers in fascial apertures through which perforators run also plays a part in preventing outward blood flow.



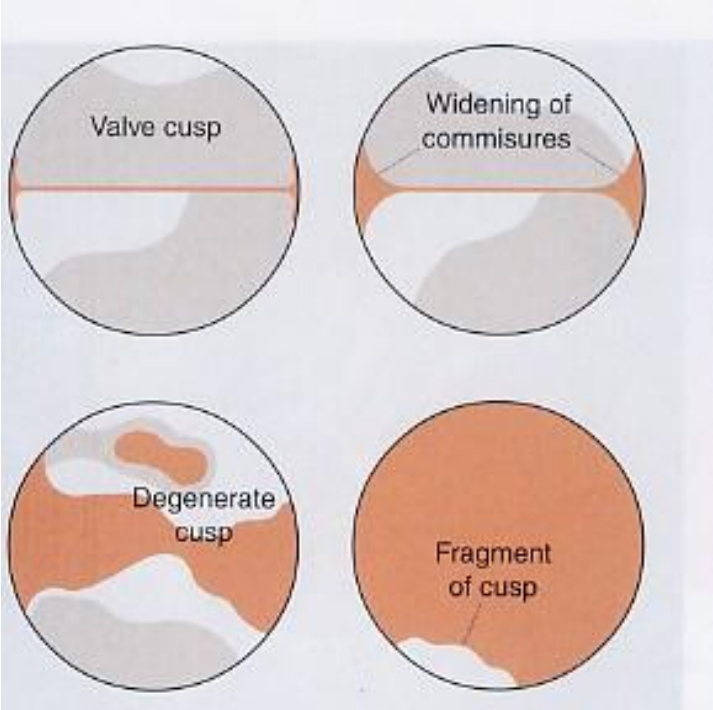
PERFORATORS OF LOWER LIMB

Morphology of vein valve

Except in common iliac veins, vena cava, portal system and cranial sinuses valves are present in most of the superficial and deep veins. Each valve is made up of two gossamer thin cusps which inspite of their delicate appearance are surprisingly strong. Valves allow blood flow only towards heart.

Their failure has documented to be one of the most important in the etiopathogenesis of varicose veins.

The mechanism that cause the superficial vein valves to fail have not been fully established.



STAGES OF DEVELOPMENT OF VENOUS VALVULAR INCOMPETEN CE

VENOUS PATHOPHYSIOLOGY

Blood flows into the leg because it is pumped by the heart along the arteries. By the time it emerges from the capillaries, it is at a low pressure (about 20mmHg), but this is enough for the blood to return to the heart. Blood from the muscles of the leg returns through the deep veins. Blood from the skin and superficial tissues external to the deep fascia drains via the long and short saphenous veins-SFJ and SPJ and communicating veins into the deep veins. Valves prevent the flow of blood from the deep to the superficial systems.

The venous pressure in the foot vein on standing is equivalent to the height of a column of blood, extending from the heart to the foot. However, the same is true of the arterial system so that on standing the arterial blood pressure at the ankle rises by 80-100mmHg, depending on the height of the person. So the blood continues to circulate, even in the absence of muscle activity. However we also have a sophisticated series of muscle pumps that act as peripheral hearts in the venous system. These are made up of the deep veins of the calf and thigh which are surrounded by muscle. In addition there is a foot pump which ejects blood from the plantar veins as pressure is placed on the foot during walking. On exercise the calf and thigh muscles contract

compressing the veins and ejecting blood towards the heart. The direction of venous blood flow is controlled by the venous valves.

The pressure within the calf compartment rises to 200-300mmHg during walking and this is more than enough to propel to the blood in the direction of the heart. During the muscle relaxation phase, the pressure within the calf falls to a low level and blood from the superficial veins flows through the perforating veins into the deep veins. The consequence of this is that the pressure in the superficial veins falls during walking. Normally the pressure in the superficial veins of the foot and ankle falls from a resting level of 80-100mmHg to about 20 mmHg.

This ability to reduce the pressure in the superficial venous systems is crucial to the health of the lower limb. Patients with damage to the veins in whom of the lower limb. Venous pressure does not fall during exercise may develop varicose eczema, skin damage and eventually leg ulceration.

PATHOLOGY OF VARICOSE VEINS

Varicose veins caused by venous insufficiency, a state of inadequate venous return in upright position and accompanied by venous hypertension may occur in following circumstances.

1. The overwhelming pumping mechanism by massive down flow in superficial veins with deficient or defective valves, as often occurs in simple varicose vein.
2. In born deficiency of deep vein valves or inherent weakness of vein walls with consequent valve failure as in valveless and weak vein syndromes. Between deficient valve and weak vein whether deficiency of valve leads to venous distension and weakening of weak vein wall leads to in-approximation of valves leading to deficiency is debated.
3. Widespread impairment of musculovenous pumping mechanism during active venous thrombosis as in acute deep vein thrombosis.
4. Obstruction or deformity in the main venous conduits as a consequence of venous thrombosis as in post thrombotic syndrome.

5. Loss of deep vein valve competence or replacement of deep veins by enlarged, valveless, collateral veins as occurs in post thrombotic states.
6. Prolonged inactivity of the muscles with the limbs in dependent position as in paralysis or disease states inhibiting use of muscles. However the blood flow through muscles will be correspondingly reduced and venous hypertension may not be too severe.
7. Arterio venous fistula direct arterial inflow to the venous side can cause venotensive changes and varicose veins.

Aggravating factors:

1) **Pregnancy** – many varicose veins appear in pregnancy and some persist. Likely cause is dilating effect of estrogen upon vein walls aggravating already existing valve incompetence.
2) **Hormonal** – women are more prone then men in a ratio of 2:1 due to hormonal influence probably. This is supported by the fact that woman's varicose veins are always more troublesome and prominent just before menstruction.
3) **Prolonged standing** – Jobs involving prolonged standing have been shown to increase the likelihood of varicose vein and incidence increases with age.

CLINICAL PATTERNS

i. Great saphenous vein area alone

ii. Small saphenous vein area alone

iii. **Mixed pattern** of both the above – either both system are incompetent or one incompetent system may cross over to another system.

iv. **Unusual source pattern** – Sources may be pelvic vein, ovarian vein or testicular vein leading to incompetence down.

v. **Intricate pattern** – Incompetence may cross over from one limb to another. Incompetence in small saphenous veins with paradoxical flow, running above and emerge as incompetent great saphenous vein below.

vi. **Complex pattern –** In associated deep vein thrombosis varicosity may develop in tributaries of great saphenous vein that is acting as collateral or varicosity of opposite limb great saphenous system by cross over.

CLASSIFICATION OF VARICOSE VEINS

C – Clinical signs (grade, supplemented by “A” for asymptomatic and “S” for symptomatic presentation

E - Etiologic classification (congenital, primary, secondary)

A - Anatomic distribution (superficial, deep, or perforator, alone or in combination

P - Pathophysiologic dysfunction (reflux or obstruction, alone or in combination)

CLINCIAL CLASSIFICATION

Any limb with possible chronic venous disease is first placed into one of seven clinical classes according to the objective signs of disease.

Clinical Classification of Chronic Lower Extremity Venous Disease

Class 0	Non visible or palpable signs of venous disease
Class 1	Telangiectasia, reticular veins, malleolar flare
Class 2	Varicose veins
Class 3	Edema without skin changes
Class 4	Skin changes ascribed to venous disease (e.g., pigmentation, venous eczema, lipodermatosclerosis)

Class 5 Skin changes as defined above with healed ulceration

Class 6 Skin changes as defined above with active ulceration

Limbs in higher categories have more severe signs of chronic venous disease and may have some or all of the findings defining a less severe clinical category. Each limb is further characterized as asymptomatic (A), for example, or symptomatic (S), for example, symptoms that may be associated with telangiectatic, reticular or varicose veins include lower extremity aching, pain, and skin irritation. Therapy may alter the clinical category of chronic venous disease. Limbs should therefore be reclassified after any form of medical or surgical treatment.

ETIOLOGIC CLASSIFICATION

Venous dysfunction may be congenital, primary, or secondary. These categories are mutually exclusive. Congenital venous disorders are present at birth may not be recognized until later. The method of diagnosis of congenital abnormalities must be described. Primary venous dysfunction is defined as venous dysfunction of unknown cause but not of congenital origin. Secondary venous dysfunction denotes an acquired condition resulting in chronic venous disease, for example, deep venous thrombosis.

Etiologic Classification of Chronic Lower Extremity Venous Disease

Congenital (E_C) Cause of the chronic venous disease present since birth

Primary (Ep) Chronic venous disease of undetermined cause.

Secondary (Es) Chronic venous disease with an associated known cause (post-thrombotic, post traumatic and others)

ANATOMIC CLASSIFICATION

The anatomic site(s) of the venous disease should be described as superficial (As), deep (Ao), perforating (Ap) vein(s). One, two, or three systems may be involved in any combination. For reports requiring greater detail, the involvement of the superficial, deep, and perforating veins may be localized by use of the anatomic segments.

Segmental Localization of Chronic Lower Extremity Venous Disease

Segment No.	**Vein(s)**
Superficial Veins	
1	Telangiectasia / reticular veins
	Greater (long) saphenous vein
2	Above knee
3	Below knee
4	Lesser (short) saphenous vein
5	Nonsaphenous
Deep Veins	
6	Inferior Vena cave
	Iliac

7	Common
8	Internal
9	External
10	Pelvic : gonadal, broad ligament
	Femoral
11	Common
12	Deep
13	Superficial
14	Popliteal
15	Tibial (anterior, posterior, or peroneal)
16	Muscular (gastrointestinal, soleal, other)
Perforating Veins	
17	Thigh
18	Calf

PATHOPHYSIOLOGIC CLASSIFICATION

Clinical signs or symptoms of chronic venous disease result from reflux (R_R), obstruction (P_o), or both ($P_{R,O}$).

Pathophysiologic Classification of Chronic Lower Extremity Venous Disease

Reflux (P_R)

Obstruction (P_O), or both ($P_{R,O}$).

CLINICAL TYPES OF VARICOSE VEINS

1. **Primary or simple varicose veins:**

These occur only in the superficial veins of the lower limbs and are by far most common variety of varicose veins. Such veins have no competent valves and are subject to substantial gravitational down flow when the patient is upright and moving. This retrograde flow not allowed by valves normally leads to turbulent reversed flow beneath the valves causes varicose veins. Alternatively it is equally plausible to say that inherent weak vein walls expand in width and length so that valve cusps separate and allow reverse flow to occur. Among the weak vein wall and deficient valve which is primary process is debatable. Anyhow varicose veins are associated with retrograde flow in them and varicose veins are expression of a dynamic phenomenon and not merely static distension.

2. **Secondary varicose veins:**

Tortuosity is often seen in superficial veins carrying reversed flow as a collateral mechanism for obstruction in a neighbouring deep vein.

a. Obstruction to venous outflow

1. **Pregnancy**
2. **Fibroid**
3. **Ovarian cyst**
4. **Pelvic cancer (of cervix, uterus, ovary or rectum)**
5. **Abdominal lymphadenopathy, Ascites,**
6. **lilac vein thrombosis**
7. **Retroperitoneal fibrosis.**

b. Destruction of valve- from deep vein thrombosis.

c. High pressure flow- from arteriovenous fistula.

SYMPTOMS OF VARICOSE VEINS

1. Distress caused by unsightly and displeasing appearance

2. Aching in the vicinity of varicose veins particularly after prolonged standing.
3. A feeling of heaviness towards the end of the day.
4. In women discomfort is increased a few days before menstruation.

But there is little relationship between size of varicose vein and symptoms.

Additional symptoms of venotensive changes

1. Pruritis is early sign and scratching may lead to ulcer. It is thought to be due to haemosiderin deposition.
2. Increased discomfort – due to presence of ulcer and secondary infection.
3. Venous claudication – bursting sensation in calf after exercise in post thrombotic venous obstruction in deep veins above.

SIGNS OF VARICOSE VEINS

1. Tortuosity – dilated, tortuous, twisted, knotted or lengthened veins. It indicates reversed flow.

2. Saccules in veins - (example saphena varix) saphenous vein is too robust to become tortuous and saccule due to turbulent flow below valve cusps appear.

3. Inky blue black veins – due to stretched overlying skin.

4. Distended subdermal and intradermal venules – seen around the ankle and on foot known as corona phlebectatica. These denote increased venous pressure.

5. Hollows and grooves in the elevated limbs due to collapsed veins surrounded by induration.

SIGNS OF VENOTENSIVE CHANGES

1. **Swelling** – due to edema added with volume of overdistended veins.
2. **Induration** – diffuse fibrosis in subcutaneous tissue caused by chronic inflammatory changes and fat necrosis called **lipodermatosclerosis** causes in duration.
3. **Pigmentation** – due to accumulation of haemosiderin.
4. **Ulceration** – hypoxia caused by fibrin layer leaked from vein leads to ulceration.
5. **Eczema** - Skin becomes more pone to eczema and extremely vulnerable to any sensitising agents including antibiotic creams and bandage materials.

COMPLICATIONS OF VARICOSE VEINS.-

1. HAEMORRHAGE.

It may occur from minor trauma to the dilated vein. The bleeding may be profuse due to high pressure within the incompetent vein. Simple elevation of the leg does a lot to stop such bleeding.

2. PHLEBITIS.

This may occur spontaneously of secondary to minor trauma. Mild phlebitis may be produced by the sclerosing fluid used in the injection

treatment. In this condition various vein become extremely tender and firm. The overlying skin becomes red and oedematous. Pyrexia and malaise may be associated with.

3. ULCERATION

This is more due to deep venous thrombosis rather than various veins alone. The patients often give previous history of venous of the thrombosis suggested by painful swelling of the leg. After thrombosis has been recanalised the valves of the deep veins are irreparably damaged. The deoxygenated blood gets stagnated in the lower p[art of the leg particular on the medical side where there are plenty of perforating veins. The superficial tissue loses its vitality on certain extent and a gravitational ulcer follows either spontaneously or following minor trauma. These ulcers have ragged edges. If the ulcer is healing, a faint blue rim of advancing epithelium may be seen at the margin. Rarely malignancy can develop in the edge of a long standing venous ulcer.

4. PIGMENTATION.

5. ECZEMA.

6. LIPODERMATOSCLEROSIS

This means the skin becomes thickened, fibrosed and pigmented. This is due to high venous pressure which causes fibrin accumulation around the capillary and it also activates white cells.

7. CALCIFICATION OF VEIN

8. PERIOSTITIS in case of long-standing ulcer over the tibia.

9. EQUINES DEFORMITY

This only result from long-standing ulcer. When the patient finds that walking on the toes relieves plain, so he continues to do so and ultimately the Achilles tendon becomes shorter to cause this defect.

CLINICAL TESTS

BRODIE- TRENDELENBURG TEST

This test is performed to determine the incompetency of the sapheno-femoral valve and other communicating system. This test can be preformed in two ways. In both the methods, the patients is first placed in the recumbent position and his legs are raised to empty the veins. This may be hastened by milking the veins proximally. The sapheno-femoral junction is now compressed with the thumb of the clinician or a tourniquet is applied just below the sapheno-femoral junction and the patient is asked to stand up quick. (1) In first method, the pressure is released. If the varices fill very quick by a column of blood from above, it indicates incompetency of the sapheno-femoral valve. This is called a positive Trendelenburg test. (2) To test the communicating system, the pressure is not released but maintained for about 1 minute. Gradual filling of the veins during the period indicates incompetency of the communicating veins, mostly situated on the medial side of the lower half of the leg allowing the blood to flow from the deep to the superficial veins. This is also considered as positive Trendelenburg test and the positive tests are indications for operation.

In case of short saphenous vein same test is done by pressing the sapheno-popliteal junction.

2. TOURNIQUET TEST

It can be called a variant of Trendelenburg test. In this test the tourniquet is tied round the thigh or the leg at different levels after the superficial veins have been made empty by raising the leg in recumbent position. The patient is now asked to stand up. If the veins above the tourniquet fill up and those below it remain collapsed, it indicates presence of incompetent communicating vein above the tourniquet. Similarly if the veins below the tourniquet fill rapidly whereas veins above the tourniquet remain empty, the incompetent communication vein must be below the tourniquet. Thus by moving the tourniquet down the leg in steps one can determine the position of the incompetent communicating vein.

The number of incompetent communicating sources in the lower in the long saphenous vein is the sapheno-femoral junction (most important), the midthigh perforator, the lower-thigh perforator and the lower leg perforators on the medial side.

In case of short saphenous incompetence-application of the venous tourniquet to the upper thigh has the paradoxical effect of increasing the strength of the reflux, as shown by faster filling time. The sign which has not been described before is pathognomonic of varices of the short saphenous system. The mechanism is a very simple-application of the upper thigh tourniquet blocks off the normal internal saphenous system which is carrying

most of the superficial venous return and thus through into greater prominence the retrograde leak for the saphenous-popliteal junction following examination:- The saphenopopliteal junction is marked with a pen with the patient standing. The short saphenous vein is emptied by elevation of the leg, firm thumb pressure is applied to the ink mark. The patient is made to stand. The pressure is released and the vein will be filled immediately. For all practical purposes that there is no other incompetent perforating vein in the short saphenous system should be remembered.

3. PERTHE'S TEST

The affected lower extremity is wrapped with elastic bandage. With the elastic bandage on the patient is instructed to move around and exercise. Severe crampy pain is complained of, if there is deep vein thrombosis. Of course arterial occlusive disease should be excluded.

4. PERTHE'S TEST (MODIFIED)

This test is primarily intended to know whether the deep veins are normal or not. A tourniquet is tied round the upper part of the thigh tight enough to prevent any reflux down the vein. The patient is asked to walk quick with the tourniquet in place. If the communication and the deep veins are normal the varicose veins will shrink whereas if they are varicose veins will be more distended.

5. SCHWARTZ TEST

In a long-standing case if a tap is made on the long saphenous varicose vein in the lower part of the leg an impulse can be felt at saphenous opening with the other hand.

6. PRATT'S TEST

This test is performed to know the positions of leg perforators. First an Esmarch elastic bandage is now applied from toes to the groin. A tourniquet is then applied at the groin. This causes emptying of the various veins. The tourniquet is kept in position and the elastic bandage is taken off. The same elastic bandage is taken off. The same elastic bandage is now applied from the groin downwards. At the positions of the perforators blow outs, or visible varices can seen. These are marked with a skin pencil.

7. MORRISSEY'S COUGH IMPULSE TEST

The limb is elevated to empty the varicose veins. The limb is then put to bed and the patient is asked to cough forcibly. An expansile impulse is felt in the long saphenous vein particularly at the saphenous opening if the sapheno-femoral valve is incompetent. Similarly a bruit may be heard on ausculation.

8. FEGANS METHOD TO INDICATE THE SITES OF PERFORATORS

In standing posture the places of excessive bulges within the variocosities are marked. The patient now lies down. The affected limb is elevated to empty the varicosed veins. The examiner palpates along the line of the marked varicosities carefully and finds out gaps or pits in the deep fascia which transmit the incompetent perforators.

9. One should look for pitting oedema or thickening, redness or tenderness at the lower part of leg. These changes are due to chronic venous hypertension following deep vein thrombosis. Sometimes a progressive sclerosis of the skin an subcutaneous tissue may occur due to fibrin deposition, tissue death and scarring. This is known as lipodermatosclerosis and is also due to chronic venous hypertension. This may follow formation of venous ulcer.

PERCUSSION

If the most prominent parts of the varicose veins are tapped, an impulse can be felt by the finger at the saphenous opening. This is known as Schwartz test. Sometimes the percussion wave can be transmitted from above downwards and this will imp[ly absent ot incompetent valves between the tapping finger and the palpating finger.

AUSCULTATION

The importance of auscultation is limited to the arteriovenous fistula, where a continuous machinery murmur may be heard.

INVESTIGATIONS

1. Doppler flow meter and ultrasonography

Doppler flowmeter and ultrasonography can detect direction of flow. In this instrument a piezoelectric crystal in the probe emits a continuous ultrasound signal, this reflected back by red cells to a receiving crystal. According to the speed and direction of movements of red cells there is Doppler shift in the phase of the signal which is recognized by the machine and made apparent audibly, by a needle gauge, by LED display or by oscillograph. Signal generated represents the speed of flow not the volume. So strong signal may be caused by high speed jet of the blood leaking through valve cusps and may not indicate severity of incompetence. In tortuous veins confused signals may be obtained due to close proximity of conflicting flows or from uncertainty about orientation. These difficulties may be resolved by asking the patient to cough in order to create a spurt of flow downwards or deliberately pressing vein down to cause a small peak. Deep veins may also be studied but not with the certainty as with superficial vein.

2. Measurement of venous pressure in leg or foot

The fall in venous pressure caused by exercise in upright position is regained normally in 20 seconds. In superficial or deep vein incompetence the refilling is quick as there is massive reflux. This may be measured directly be inserting a plastic cannula attached to electronic transducer into a ankle or foot vein or indirectly by photo plethysmography.

Plethysmography reads change in limb volume which is closely parallel to venous pressure.

a. **Fluid plethysmography** – Leg or foot immersed in water in a fixed chamber and amount of fluid displaced measured. Artifacts caused by weight of fluid and limitation of movements for patients are disadvantage.
b. **Air plethysmography** – Leg is surrounded by air filled PVC chamber and amount of air entering or leaving is measured. Temperature variation is main disadvantage.
c. **Electrical impedance plethysmography** – Estimates volume change with alteration in electrical resitance.
d. **Strain gauge plethysmography** – Limb is surrounded by slender elastic tube containing mercury or similar electrically conducting fluid, the resistance of which will vary as the diameter of elastic tube changes with volume change of limb. Its main value is in gauging recovery time.

e. **Photoplethysmography** – This photoelectrically estimates number of R.B.C. in skin capillary bed underlying transducer. A light emitting diode which gives off heat to cause artifact illuminates the capillary bed with infrared light. This is absorbed by hemoglobin in the red cells and the amount transmitted back to sensor is measured. It measures congestion in skin capillaries and thereby venous pressure.

Maximum venous outflow: Strain gauge plethysmography may be used to measure the maximal speed at which venous blood can leave a limb and from this restriction in venous flow is estimated. In this reduction in venous congestion after release of pneumatic cuff is measured. Normally 90% of fall in volume occurs in first 3 seconds.

3. Functional phlebography

In this venous system is visualised in addition to finding out superficial and deep vein flow in response to exercise and patency of valves.

Non irritating osmolar radio opaque medium such as IOHEXOL is used and introduction of image intensifier allows prolonged viewing without exposing patient to excessive radiation. More that one view in different rotation is necessary to assess plane or vein. In the foot down position deep

veins are gradually visualized and by introducing few exercise movements flow pattern is studied.

Swill test – patient is put on head down position so that veins slightly deflate and sudden head and elevation to near vertical plane demonstrates valves.

4. Ultrasonographic imaging with duplex and colour flow scanning

Creation of image by pulsed beam ultrasound scanning combined with Doppler flow facility is Duplex scanning.

This technique involves use of high resolution B-mode ultrasound imaging and Doppler ultrasound to obtain images of veins and simultaneously measure flow in these vessels.

Direction of flow that is blue towards heart and red away from the heart is added facility in Triplex scanning.

TREATMENT

Non Operative

Compression Therapy

This is the mainstay in the management of CVI. Compression can be achieved using a variety of techniques including elastic compression stockings, paste Gauze boots – Unna's boot, multilayer elastic wraps or pneumatic compression devices. A number of physiologic alternative have been observed with compression.

These include

1. Improvement in skin microcirculation
2. Increase in subcutaneous pressure which counters transcapillary leak
3. With edema reduction, cutaneous metabolism may improve due to enhanced diffusion of oxygen and often nutrients.

Compression therapy is mostly achieved with graded elastic compressions stockings. Below knee 30-40 mmHg elastic compression stocking resulted in 93% healing in 5 months. Ulcer recurrence was less in patients who were compliant.

Disadvantages

1. Compliance
2. Hyper sensitivity
3. Difficulty in application

Another method of compression is Unna's boot. A typical Unna's boot a rolled gauze impregnated with calamine, zinc oxide, Glycerine, sorbitol, gelatin and magnesium aluminium silicate is first applied with graded compression from forefoot to just below knee.

The Next layer consist of 4 inch wide continuous gauze dressing followed by an outer layer of elastic wrap also applied with graded compression. The bandage becomes stiff after drying and the rigidily may aid in preventing edema foundation.

The medium time of healing was 9 weeks.

Disadvantages

1 Contact dermatitis

2. Bulkiness

Sclerotherapy

This technique is useful in veins less than 3mm Diameter. Acts by destroying venous endothelium.

Indications for sclerotherapy

Optimal Indications

Telangiectasias

Isolated varicosities

Below knew variocosities (In absence of gross saphenous reflux)

Recurrent varicosities (In absence of gross saphenous reflux)

Less than optimal indications

Symptomatic reflux

In the aged and infirm

In non-surgical candidates

Questionable indications

Greater saphenous reflux

Lesser saphenous reflux

Large varicosities

Contra indications for sclerotherpay

Allergy to sclerosant

Presence of arterial occlusive disease

Patient immobility

Presence of uncontrolled malignant tumour

Acute thrombophlebitis

Huge varicosities with large communication to deep veins

Agents are

11.7 – 23.4% Hypertonic saline saline

.125 - .25% Sodium Tetradecyl sulphate

.5% Polidocanol

Larges varicose veins require higher concentration

23.4% Hypertonic saline saline

.5 - .75% Sodium Tetradecyl sulphate

.75 – 1.0% Polidocanol

Methods: Veins to be treated are identified and marked. Intra arterial accidental injection should be avoided. Preparations to manage anaphylaxis should be ready. Needles of syringes containing 0.5ml sclerosant introduced into veins and position confirmed. Limb is elevated and veins are emptied. Sclerosant injected and compression applied compression maintained for 3 weeks. Exercise advised during this period to avoid deep vein thrombosis.

Complications

a. Allergic reaction
b. Pigmentation
c. Thrombophlebitis
d. DVT
e. Skin necrosis

SURGICAL TREATMENT

Essential principle is that source of incompetence, incompetent path way and reentry point into deep vein system i.e., incompetent perforator, all should be tackled.

First opportunity for surgery should be utilised for complete removal because postoperative sclerotherapy for remaining varicosities is tiresome for patient.

In patients with symptomatic GSV Refeux, GSV should be removal small incision placed medially in the groin, and just below knee and GSV stripped.

Sub optimal treatment may cause recurrence which may cause more morbidity and treatment difficult because of fractured anatomy.

TREDELENBURG'S SURGERY

Procedure for Great saphenous varicosity

Skin incision 2cm below and lateral to pubic tubercle, great saphenous vein termination identified and all tributaries at termination ligated and divided. Flush ligation of saphenofemoral junction done.

A test for incompetent pathway by injecting saline into distal tributaries thro’ cannula attached to 20ml syringe should be done, since stripping out great saphenous vein leaving behind incompetent tributary e.g. anterolateral branch may lead to recurrence. Resistance while injecting denotes competent valve.

Saphenous vein exposed below knee, saphenous nerve preserved and Mayo’s stripper introduced from below, vein tied around stripper and stripping done. Compression applied immediately to avoid hematoma.

Stripping upto ankle is not necessary because usually the posterior arch vein is at fault and procedure may injure saphenous nerve.

Phlebectomy of remaining varicosities done by stab and avulse technique.

Small saphenous vein varicosity

Preoperative assessment of saphenopopliteal opening is necessary during investigation. By transverse incision at the level of saphenopopliteal junction small saphenous vein identified without doubt. Stripper introduced from the position where lowest varicosity arise because stripping from lower leg may injure sural nerve.

Stripping completed and compression applied. Other varicosities removed by multiple phelebectomies.

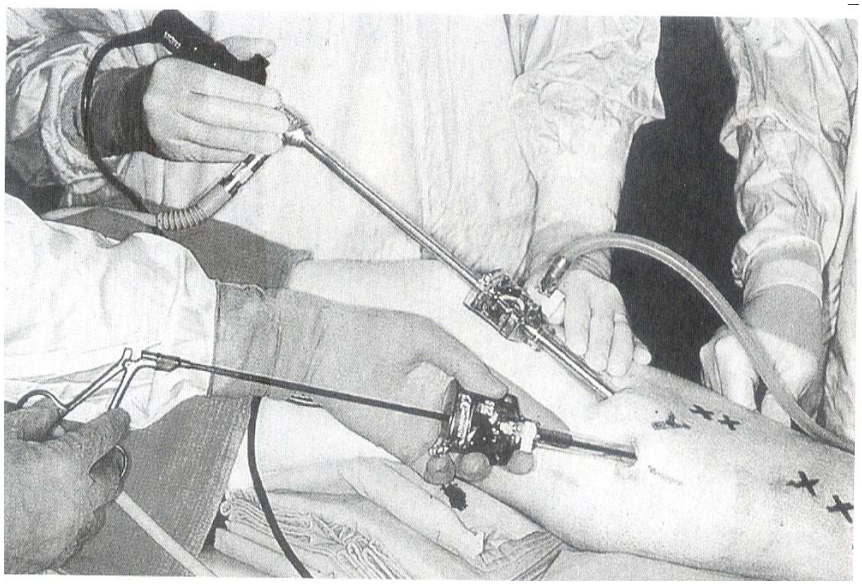
Perforaton vein ligation

Incompetence of perforating veins connecting the superficial and deep venous systems of lower extremities was best handled by LINTONS ligation.

SEPS

A newer minimally invasive technique termed as SUBFACIAL ENDOSCOPIC PERFORATOR VEIN SURGERY (SEPS) has evolved with the improvement in endoscopic equipment.

DUS is performed preoperatively in patients undergoing SEPS to document deep vein competence and to identify perforating veins in the perforating compartment.



Procedure :-

The patients is posterior on the operating table with affected leg elevated at 45^0-60^0. An esmarch bandage and a thigh tourniquet are used to exsanguinate the limb. The knee is then flexed and two small incisions are made in the proximal medical leg away from areas of maximal induration at the ankle.

Laproscopic trocars are the positioned and the subfacial dissection is performed with a combination of blunt and sharp dissection. Carbon dioxide is then used to insufflate the subsacial space. The thigh tourniquet is inflated to prevent air embolism. The perforator are them identified and doubly clipped the divided. After completion, the leg is wrapped in a compression bandage for 5 days past operatively.

TREATMENT OF VENOTENSIVE CHANGES AND ULCERATION

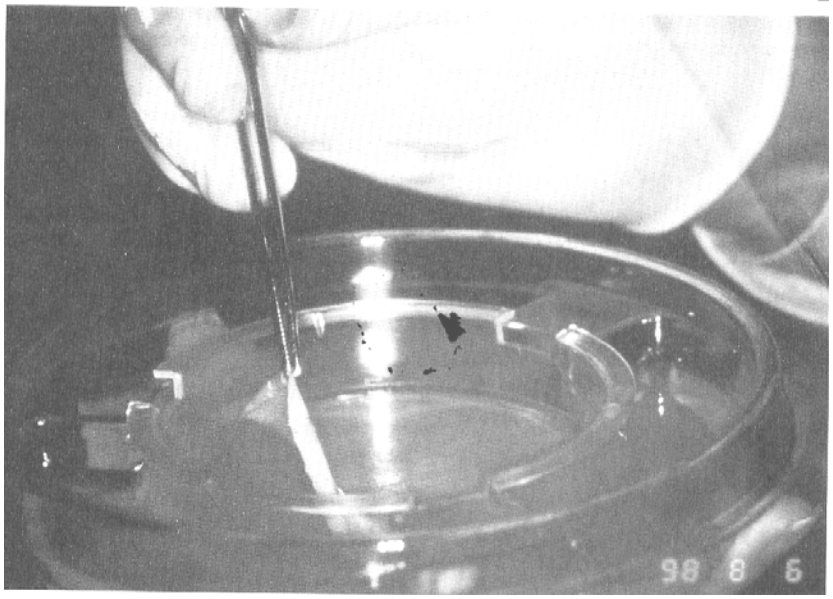
In patients who have venous ulceration due to superficial venous incompetence alone, varicose vein surgery is effective in producing ulcer healing in those patients who are fit enough to undergo this treatment. It is not necessary to delay surgery until the ulcer has healed. The ulcer is covered by a dressing during surgery and prophylactic antibiotics are given to prevent infection of the wounds with any bacteria. Ulcers managed in this way usually heal rapidly (with in 4 weeks) following surgery.

Skin substitutes:

Bio engineered skin ranges in composition from acellular skin substitutes to partial living skin substitutes. They also served as delivery vehicles for growth factors and cytokines.

APLIGRAF is a commercially available bilayered living skin construct that closely approximates human skin. It contains a protective stratum corneum and a keratiuocyte containing epidermis overlying a dermis consisting of DERMAL FIBROBLARTS in a collagen matrix. Thickness is 0.5mm to 10.mm and is supplied as a disk of living tissue in agarose gel nutrient medium. It must be used with in 5 days. Ulcer heals at 6 month disadvantages

1. Rejection
2. Hyper sensitivity



APLIGRAF

Recurrent varicose vein

Recurrence may be due to

a. Persistent varicose vein caused by original misdiagnosis and incorrect identification of pathway of incompetence and inadequate removal of pathway.
b. True recurrence: Varicose veins first disappear and then reappear due to following reasons
    i) Inadequate removal of source.
    ii) Anatomical reconnection through existing tributaries or non anatomical reconnection by neovascularisation.
    iii) Latent second source becoming acting and causing varicosity.

iv) Primary valve deficiency or weak vein syndrome.

Treatment

Compression sclerotherapy of residual varicosities and minor recurrences.

Major recurrences managed by surgical excision of complete pathway.

Prevention by conservative management with alternating period of leg elevation and compression bandages.

Complications of varicose veins and management

Haemorrhage

- Common in elderly around foot, ankle and lower leg.
- Immediate management by foot and elevation and compressive bandages.
- Completion treatment with either sclerotherapy or surgery.

Thrombophlebitis

Conservative management is with anti inflammatory drugs, foot and elevation after checking peripheral pulses and compression bandage.

Surgical excision is curative and if it is not possible evacuation of clot done.

Syndromes of valve deficiency and weak vein walls

- Incidence is 8%
- Spectrum of valve deficiency includes well defined states at one end with incompetent superficial veins to absent valves in deep and superficial veins.
- No history of a previous deep vein thrombosis.
- Multiple incompetent valve sources.
- Selective trendelenburg test does not control enlarged superficial veins due to incompetent deep veins.
- Severe venotensive changes.
- Doppler flowmetry shows surge back and forth with little purposeful movement on either direction.
- Photoplethysmography shows short recovery time not improved by manipulation of superficial veins.
- Phlebography – no evidence of deep vein thrombosis, widely open, few functioning valves.
- Ultrasonography shows reflux and lack of valves.

Klippel – Trenaunay syndrome

Extensive venous abnormality described above often associated with overgrowth in bones to give increased length in limbs. No demonstrable congenital arterio venous fistula.

Treatment

If deep veins are widely open and valveless it is permissible to remove valveless superficial veins. In borderline cases this reduction in overload may improve pumping mechanism.

Conservative line of management with periodical alternate foot and elevation, compression bandages indicated for sever cases and for weak vein syndromes.

Surgical restoration of valve may be tried

1. Transposition of deep vein to a neighboring vein that is well valved.
2. Transplantation of suitable venous valve taken elsewhere from body for example. Taheri operation – transplanting brachial or opposite saphenous valve to upper popliteal.
3. Repair of prolapsing valve cusps by valvuloplasty – Kirstner operation.
4. Use of tendon or silastic sling as substitute to valve – Psathakis.

POST THROMBOTIC SYNDROME

Post thrombotic syndrome is associated with valve destruction and collateral flow formation.

Clinical patterns

i) **Tibiopopliteal** – Deep veins below knee involved associated with refluxing perforators and venotensive complications.

ii) **Femoropopliteal –** Common and more easily recognized with swelling below knee, superficial collaterals and venous hypertension.

iii) **Iliac –** Left common iliac veins is vulnerable because of compression by right common iliac artery. Associated with development of collateral form left to right and varicosities.

iv) **Ileofemoral** – Severe venous hypertension and heavy reflux in recanalised veins.

Treatment

Conservative management

i) Elevation of foot above the horizontal level

ii) Improving pumping mechanism in the form of inelastic external support with 7.5 cm cotton bandage impregnated with zinc oxide, applied from toes upto knee.

This allows return of blood through collateral without occluding them and at the same time prevents pooling of blood in these veins.

Elastic compression which gives sustained 20 to 40 mm of Hg pressure may be a significant handicap to arterial perfusion.

Graded elastic compression

- Class I gives 14-17 mm of Hg
- Class II gives 18-24 mm of Hg
- Class III gives 25-35 mm of Hg

Surgical management

Surgical reconstruction of veins and valves may be tried.

i) **Palma operation:** Opposite side great saphenous vein is mobilised, divided at lower thigh, and anastamosed to common femoral or profounda femoris vein of opposite side. This acts as additional collateral. Alternatively PTFE vascular graft with external supporting rings may be used between affected femoral vein and opposite lower iliac vein, supported by temporary arteriovenous fistula.

ii) **May-Husni operation:** Popliteal to upper femoral by passing to relieve obstruction at lower or mid thigh. This involves anastamosing long saphenous vein above and below supported by temporary arterio venous fistula.

iii) Restoration of valve function as in valveless vein disorder may be tried in certain conditions.

A-V fistula

Congenital multiple arterio venous fistula

(Parkers – Weber syndrome)

Associated with limb lengthening, cardiac failure and venous hypertension.

Treatment

- Conservative management with limb elevation and elastic bandage.
- If localised transluminal occlusion may be tried.

OBSERVATION

1) Incidence of varicose veins is more common in age group 30-40 years.
2) Incidence of varicose veins is male : female 60:15.
3) Incidence of varicose veins in patients with occupation requiring long period of erect posture such as tea shop workers, hotel workers, traffic police etc. versus others was 60:15.
    a. Primary varicose veins – 68
    b. Secondary varicose veins
        i. Post thrombotic – 5
        ii. Arteriovenous fistula – 2

4) Incidence of unusual pattern is associated with secondary varicose veins.
5) Bilateral disease was seen in 18 patients.
6) Most of patients admitted were with venotensive changes.
7) All patients who underwent investigations showed positive findings.
8) Treatment given was mostly surgical.

DISCUSSION

Age incidence

In this study it was found that incidence of varicose veins and its complications are increasing steadily with age.

5 patients were found in the age group 10-20 years. 14 patients were found in the age group 20-30 years. 30 Patients were found in the age group 30-40. 16 patients were found in the age group 40-50 years. 6 patients were found in the age group 50-60 years. 4 patients were found in the age group 60-70 years.

Youngest patient in the study was 18 years old and eldest patient was 70 years. This pattern denotes that erect posture takes its own time in

aggravating the incidence of varicose veins even though the defect in value is congenital. It was found that the age group of 30-40 years had the maximum number of patients.

SEX INCIDENCE

Eventhough male: female ratio is 1:2 according to textbooks, in this study of 75 patients only 15 were female patients. 60 patients were male patients. This may be due to the fact that females may not be engaged frequently in occupation demanding long periods of erect posture. Out of fifteen, 11 patients had the appearance of varicose veins during their first pregnancy and rest of 4, deep vein thrombosis was the cause.

ANALYSIS OF OCCUPATION

In this expect 10 patients all other 65 patients had occupation demanding long period of erect posture.

- Tea shop Workers 25%
- Hotel Workers 15%
- Police 10%
- Agriculture Workers 10%
- Vendor 5%
- Tailor 5%
- Others 5%

ANALYSIS OF ETIOLOGICAL FACTORS

1. Primary varicose veins	:	68
2. Secondary varicose veins	:	7
i) Post thrombotic	:	5
ii) A-V Fisula	:	2

Most of the varicose veins were found to be of primary type i.e in 68 patients.

5 patients were found to be having associated deep vein block or post thrombotic destruction of deep vein valves.

Most of them had collateral circulation in their superficial veins. In these patients surgery will be dangerous and may aggravate the problem. Two patients had post traumatic arterio venous fistula as the cause of varicose veins.

CLINICAL PATTERN OF VARICOSE VEINS

Clinical pattern of varicose veins indicated to some extent, underlying pathology in this study. Patients with simple pattern or mixed pattern of varicose vein had defective valves in investigations patients with unusual pattern like associated abdominal varicosities, varicosities over lateral aspect of thigh and varicosities over patella had primary pathology like DVT or AV fistula. The varicosity was found in near by veins, not in standard great or small saphenous vein areas.

- Great saphenous vein was involved in 59 patients
- Short saphenous vein was involved in 7 patients

- Mixed pattern seen in 4 patients
- Unusual pattern seen in 5 patients

ANALYSIS OF LIMBS AFFECTED BY VARICOSE VEINS

Unilateral Varicose veins are seen in 57 patients. Right lower limb was affected in 18 patients. Left lower limb was affected in 39 patients. Bilateral Varicose veins seen in 18 patients.

- Rt lower limb : 18 patients
- Lt lower limb : 39 patients
- B/L lower limb : 18 patients

ANALYSIS OF SYMPTOMS

Commonest presenting complaint in the study was presence of dilated vein on the legs. It was seen in 62%.

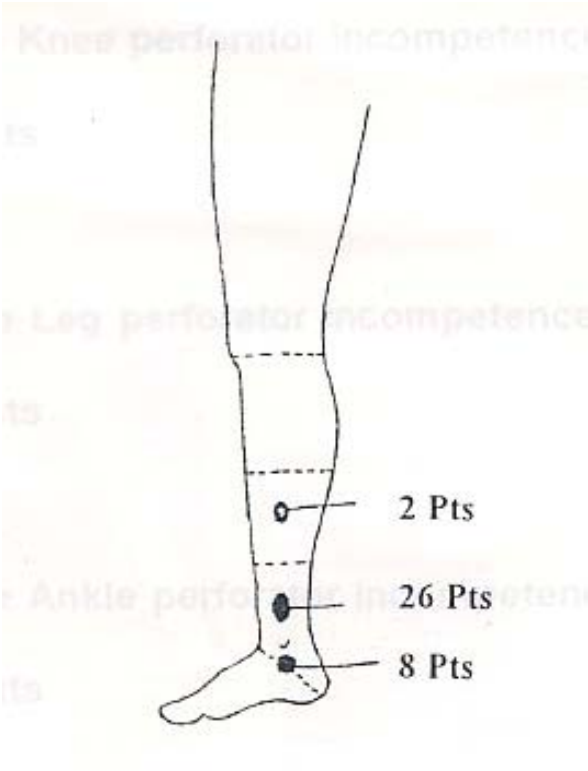
Other symptoms were

- Pain 45%
- Pigmentation 56%
- Eczema 14%
- Ulceration 36%

SITE OF ULCERATION IN VARICOSE VEINS & TREATMENT

The site of ulceration was studied in patients presenting with varicose ulcers was analysed. The commonest site of ulceration was found to be in medial side of lower 1/3 of leg. The distributions are shown in the diagram below .

In some patients with small venous ulcer healed spontaneously with trendelenburg's surgery for GSV. In patients with still persisting ulceration, SSG was done in the second sitting.



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SITES OF VENOUS ULCERS

Treatment modality

Commonest surgery performed for varicose veins in the study was trendelenburg's operation . In patients with negative Trendelenburg's test multiple stab ligation was done . In 10 patients stripping alone was done . In 6 patients with saphenopopliteal incompetence , saphenopopliteal disconnection alone was done. In 11 patiens with reticular veins – sclerotherapy was done.

SUMMARY

Varicose veins have varied etiology. Some of which can be better managed with surgical intervention and some others may be managed conservatively since surgery will aggravate the problem.

First opportunity for surgery must be utilized effectively since management of recurrent varicose veins is difficult due to fractured anatomy.

So detailed clinical examination, finding out all refluxing sources, mapping out all defective pathways with reverse flow and reentry point into deep veins is essential for optimum surgical management. All these things will be more clear with investigations.

Making the patient to learn to keep his limbs without ulcer and other venotensive changes by periodical lower limb elevation is the most important aspect of management.

Professional rehabilitation of patients is essential frequently.

PIGMENTATION AND ECZEMA IN A PATIENT WITH VARICOSE VEIN

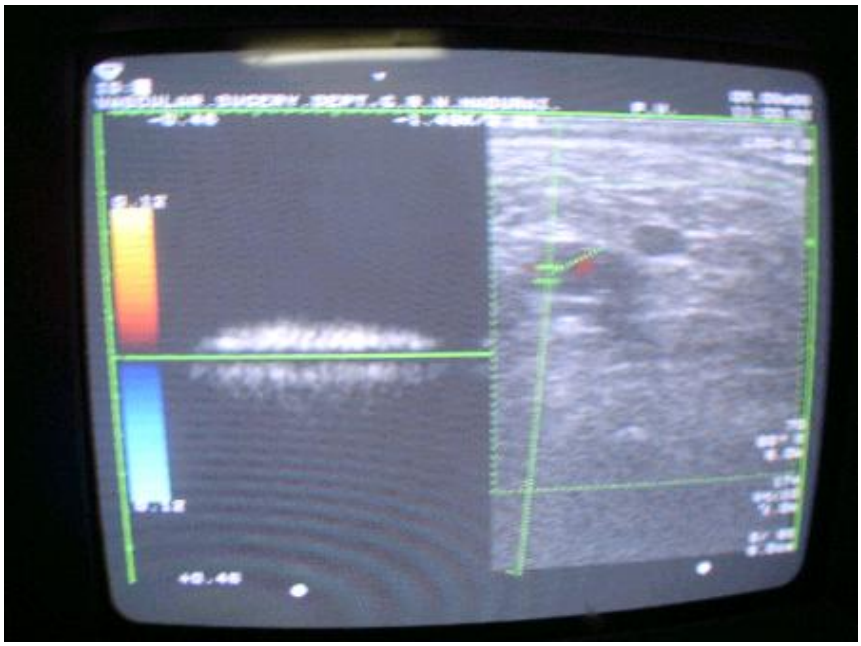
VENOUS ULCERS

PICTURE SHOWING BILATERAL VARICOSE VEINS

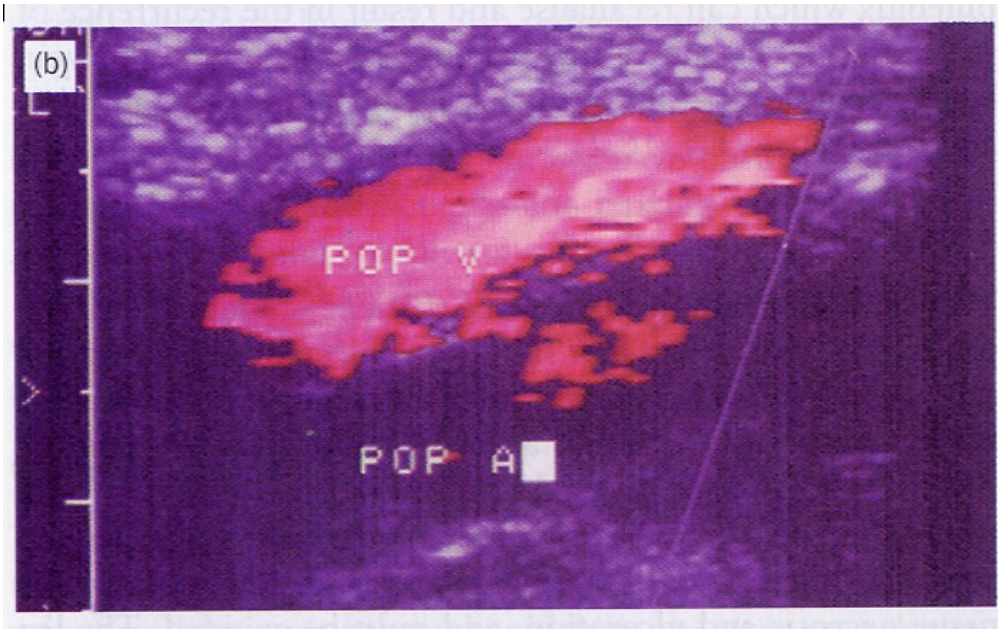
GREAT SAPHENOUS VEIN VARICOSITY

SHORT SAPHENOUS VEIN VARICOSITY

DOPPLER STUDY IN A NORMAL GSV

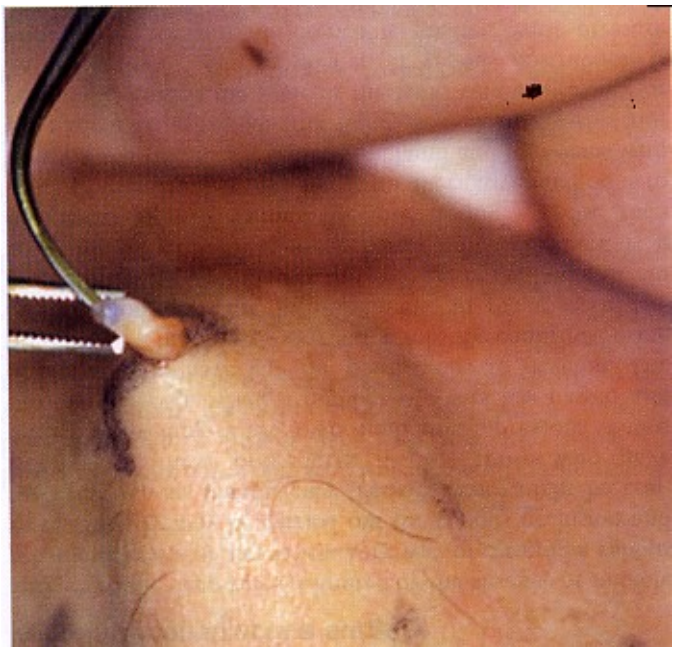


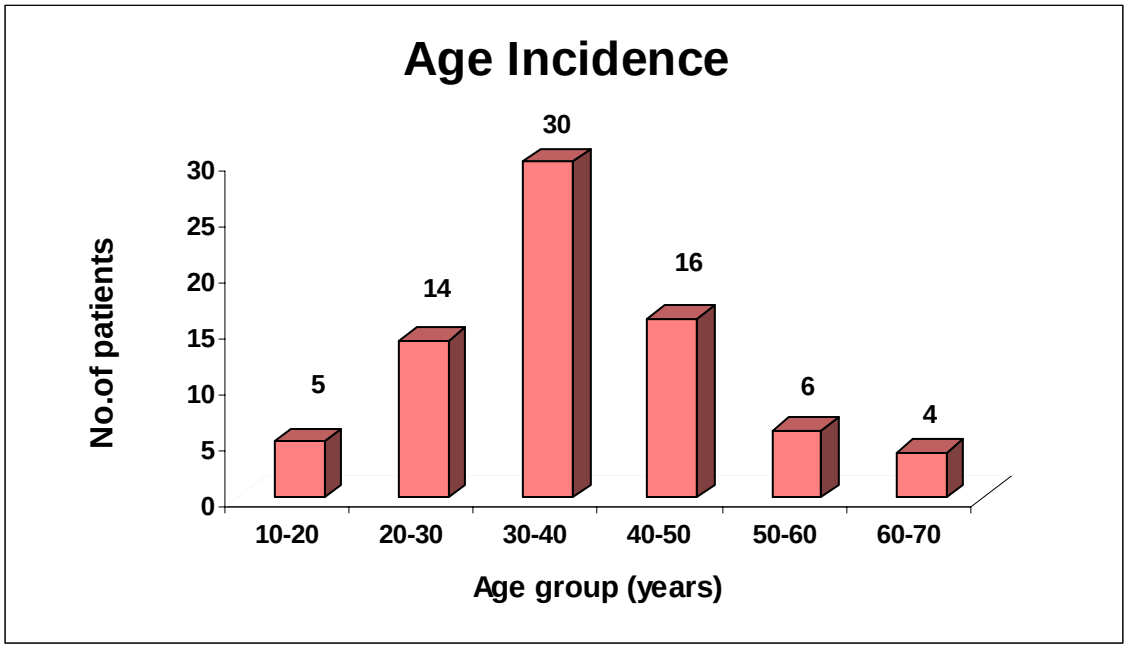
DOPPLER SHOWING VENOUS REFLEX

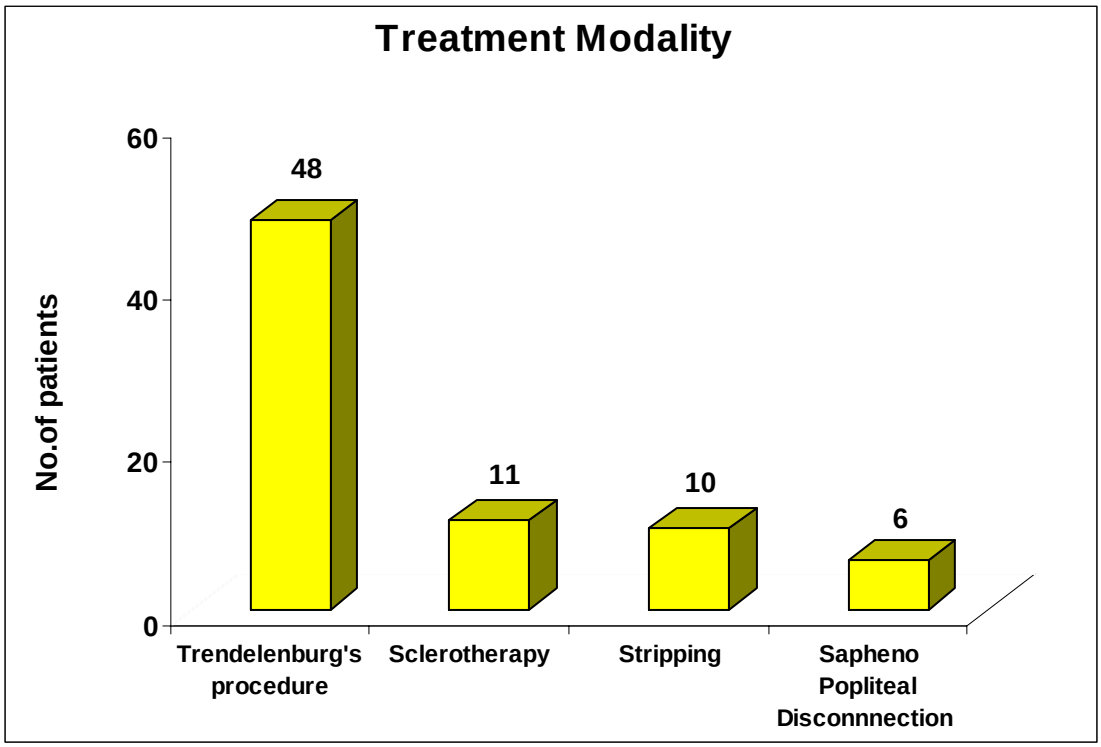


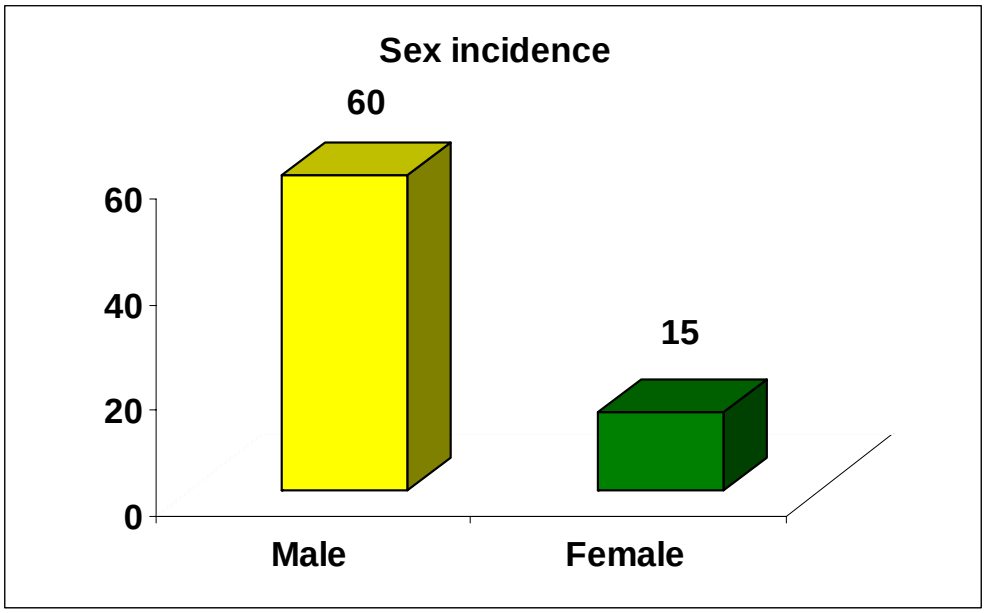
SCLEROTHERAPY FOR VARICOSE VENIS

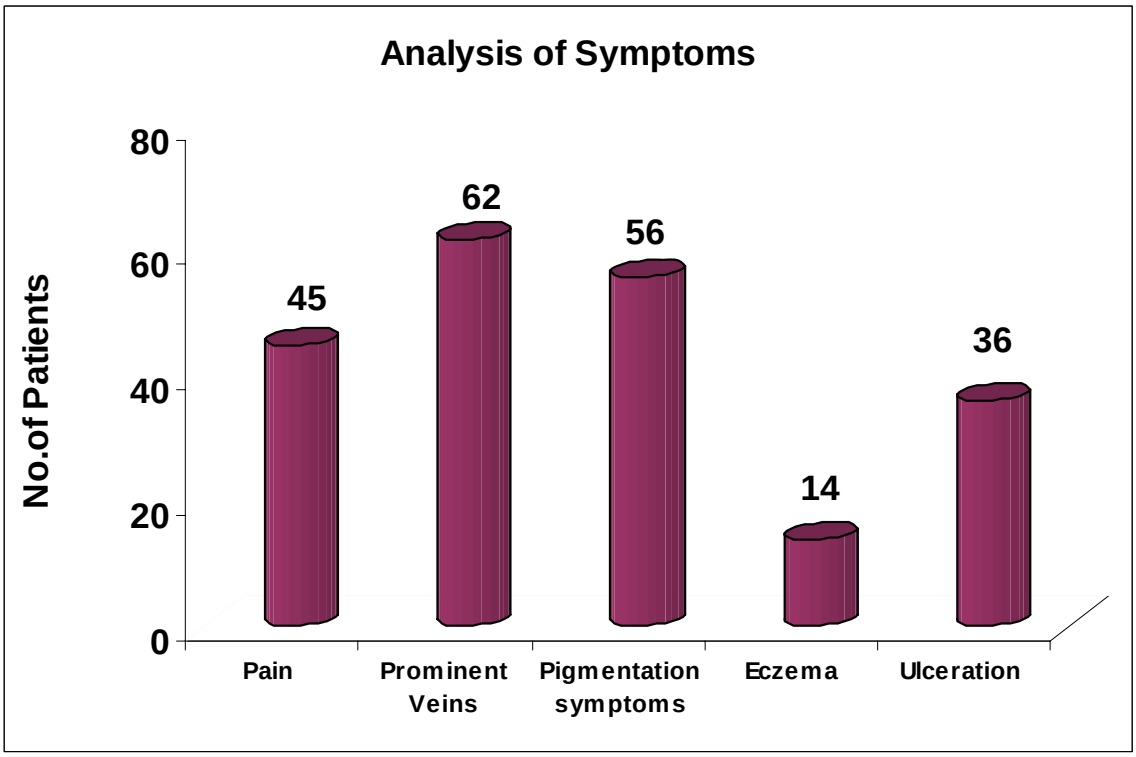
HOOK PHLEBECTOMY

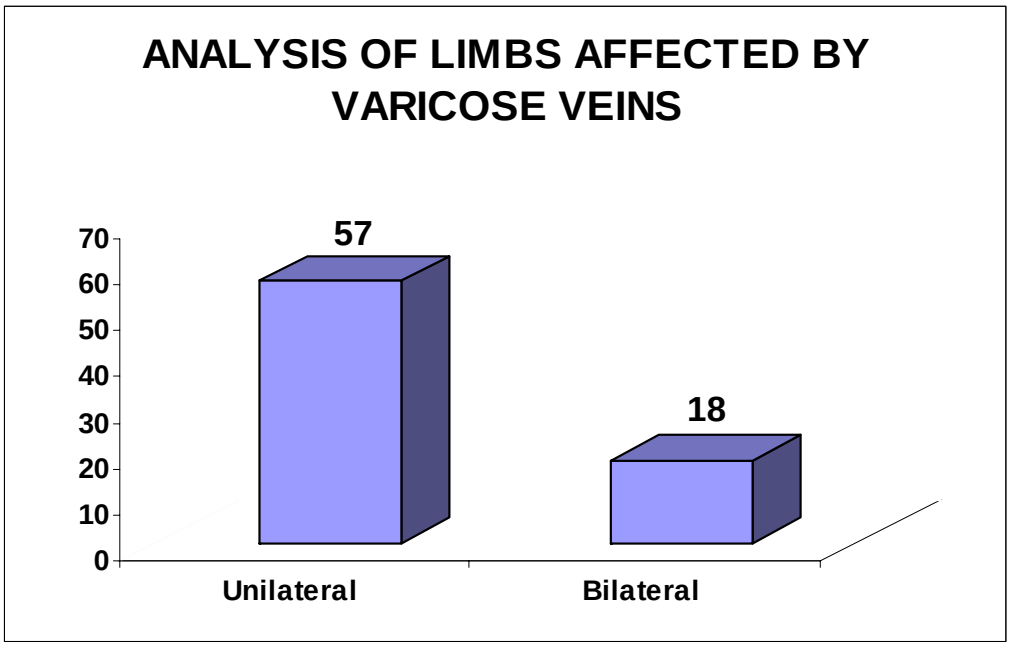


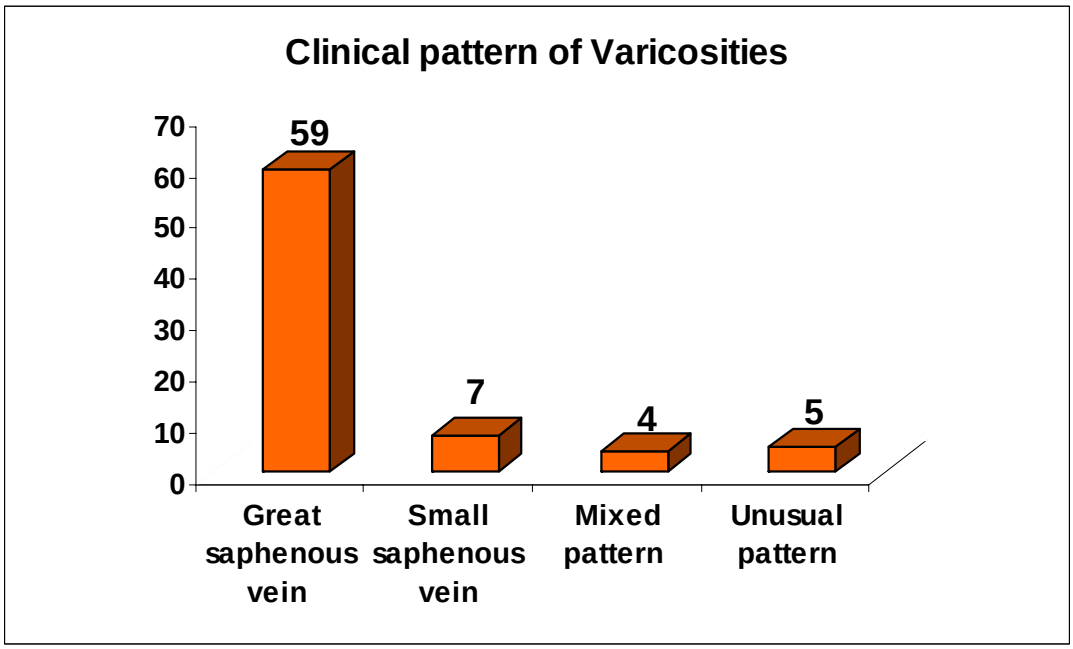

Age Incidence
No.of patients
30
25
20
15
10
5
0
5
14
30
16
6
4
10-20
20-30
30-40
40-50
50-60
60-70
Age group (years)

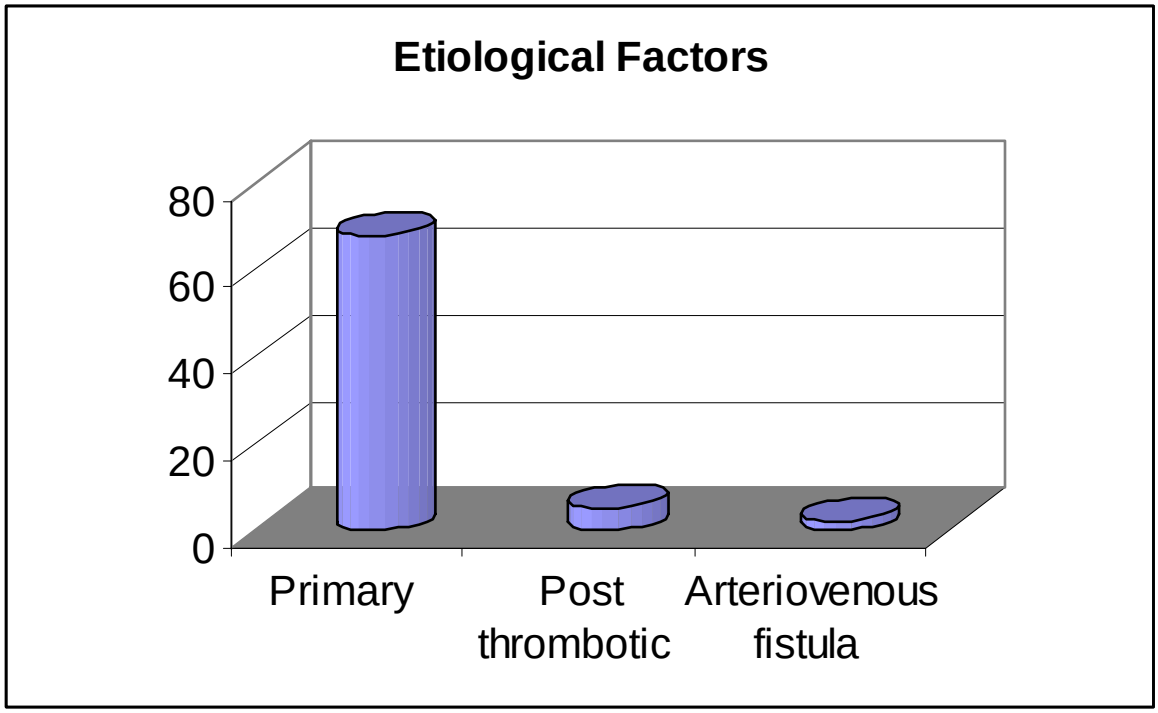

Treatment Modality
No.of patients
60
40
20
0
48
11
10
6
Trendelenburg's procedure
Sclerotherapy
Stripping
Sapheno Popliteal Disconnnection

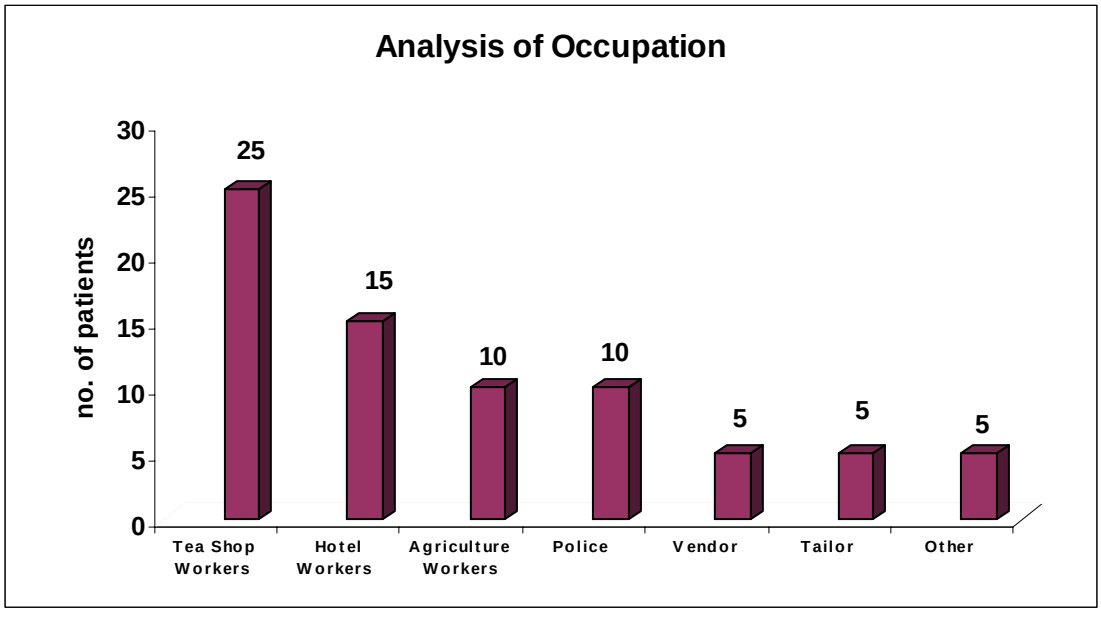

Sex incidence
60
15
60
40
20
0
Male
Female


Analysis of Symptoms
No.of Patients
80
60
40
20
0
45
62
56
14
36
Pain
Prominent Veins
Pigmentation symptoms
Eczema
Ulceration


ANALYSIS OF LIMBS AFFECTED BY VARICOSE VEINS
70
60
50
40
30
20
10
0
57
18
Unilateral
Bilateral


Clinical pattern of Varicosities
70
60
50
40
30
20
10
0
59
7
4
5
Great saphenous vein
Small saphenous vein
Mixed pattern
Unusual pattern


Etiological Factors
80
60
40
20
0
Primary
Post thrombotic
Arteriovenous fistula


Analysis of Occupation
no. of patients
30
25
20
15
10
5
0
25
15
10
10
5
5
5
Tea Shop Workers
Hotel Workers
Agriculture Workers
Police
Vendor
Tailor
Other

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PROFORMA

1. Name : Age/Sex :

2. Occupation : Income : I.P.No :

3. Address :

4. D.O.A. : D.O.S : D.O.D

5. History of present illness :

i) Duration of dilated veins : Months / Years

ii) Side : **Rt. Lower limb**
Lt. Lower limb
Bilateral

iii) Pigmentation : ☐

iv) Eczema : ☐

v) Ulceration : ☐

vi) H/o Swollen legs : ☐

vii) H/o deformity of legs : ☐

viii) Previous history of immobilisation :

Previous H/o Surgery ☐

Fracture ☐

Stroke ☐

Ch. Medical illness ☐

xi) H/o malignancy : ☐

☐

x) H/o drug intake :

xi) H/o dehydrating illness

requiring intravenous fluids : ☐

6) Past history :

1) DM 2) HT 3) PT

4) Previous H/o similar illness 5) Others

7) Personal history :

Mixed diet Alcoholic Smoker

8) Family History :

9) Examination :

1) General :

I. Nutrition :

II. Anaemia :

III. Pedal edema :

IV. Jaundice :

V. Fever :

VI. Others :

2) Local

Anterior		Posterior	
Rt	Lt	Rt	Lt

1. System involved : LSV/SSV/both / indeterminate

2. Saphenofemoral incompetence :

3. Saphenopopliteal incompetence :

4. Perforator incompetence :

Thigh :

Leg :

Ankle :

5. Deep Vein Patency :

6. Lipodermatosclerosis :

Pigmentation :

Eczema :

Ulcer :

Anterior aspect Medial aspect

7. Limb measurement : equal / unequal

Rt LL :

Lt LL :

8. Scars : Operative / traumatic

9. Auscultation over prominent veins :

Other system examination :

CVS :

RS :

P/A :

Investigation :

1) Hb% :

2) DC TC :

3) Urine routine :

4) X – ray chest PA :

5) Bl. Urea :

6) Bl. Urea :

7) Sr. creatinine :

8) Colour Doppler :

9) Venogram :

Provisional treatment :

Medical treatment :

Surgery :